```
     (FILE 'HOME' ENTERED AT 16:00:46 ON 07 MAY 2004)

     FILE 'REGISTRY' ENTERED AT 16:00:58 ON 07 MAY 2004
               E SIALYL LEWIS X/CN
L1           1 S E11
               SELECT RN L1 1-

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:01:59 ON 07 MAY 2004
L2         572 S E1
L3        3514 S SIALYL LEWIS X
L4        3657 S L2 OR L3
L5       76469 S HELICOBACTER
L6       37584 S H PYLORI
L7       76935 S L5 OR L6
L8          30 S L7 AND L4
L9          22 DUP REM L8 (8 DUPLICATES REMOVED)

     FILE 'REGISTRY' ENTERED AT 16:06:03 ON 07 MAY 2004
               E SIALYL LEWIS A/CN
L10          1 S E3
               SELECT RN L10 1-

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:06:54 ON 07 MAY 2004
L11        142 S E1
L12        727 S SIALYL LEWIS A
L13        763 S L11 OR L12
L14          5 S L7 AND L13
L15          5 DUP REM L14 (0 DUPLICATES REMOVED)
L16          1 S L15 NOT L9
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L9 ANSWER 1 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:20506 CAPLUS
DOCUMENT NUMBER: 140:87707
TITLE: Oligosaccharide therapeutic compositions for use in prophylaxis or treatment of diarrheas
INVENTOR(S): Angstroem, Jonas; Teneberg, Susann; Saarinen, Juhani; Satomaa, Tero; Roche, Niamh; Natunen, Jari; Miller-Podraza, Halina; Karlsson, Karl-Anders; Milh, Maan Abul
PATENT ASSIGNEE(S): Biotie Therapies Oy, Finland
SOURCE: PCT Int. Appl., 156 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004002495	A1	20040108	WO 2003-FI528	20030630

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: FI 2002-1275 A 20020628
FI 2003-564 A 20030414

AB The invention provides a therapeutic composition comprising purified fractions of compds. being or containing a pathogen-inhibiting oligosaccharide sequence for use as a medicament. The invention especially describes an oligosaccharide-containing substance or receptor binding to diarrheagenic Escherichia coli and/or zoonotic **Helicobacter** species, and use thereof in e.g. pharmaceutical, nutritional and other compns. for prophylaxis and treatment of conditions due to the presence of Escherichia coli and/or zoonotic **Helicobacter** species. The invention is also directed to the use of the receptors for diagnostics of Escherichia coli and/or zoonotic **Helicobacter** species.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:855945 CAPLUS
DOCUMENT NUMBER: 139:333092
TITLE: Compositions and methods for inhibiting microbial adhesion
INVENTOR(S): Holgersson, Jan; Lofling, Jonas
PATENT ASSIGNEE(S): Absorber, AB, Swed.
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003089450	A2	20031030	WO 2003-IB2253	20030422

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004009546 A1 20040115 US 2003-421197 20030422
PRIORITY APPLN. INFO.: US 2002-375102P P 20020422

AB The present invention provides compns. and methods for treating or

preventing 3 microbial infections. The invention is based in part on the discovery that carbohydrate epitopes that mediate microbial adhesion can be specifically expressed at high d. and by different core saccharides chains on glycoproteins, e.g. mucin-type protein and alpha glycoprotein backbones. The carbohydrate antigens, sialyl Lewis (e.g. Lea, Leb, Lex, Ley), are ligands for cell adhesion mols. The invention provides glycoprotein-Ig fusion proteins (referred to herein as "MA fusion protein or MA fusion peptides") containing multiple sialyl Lewis epitopes, that are useful in blocking (i.e., inhibiting) the adhesion interaction between a microbe (e.g. bacteria, virus or fungi) or a bacterial toxin and a cell. In one aspect, the invention provides a fusion polypeptide that includes a first polypeptide that is glycosylated by a α1,3 fucosyltransferase operably linked to a second polypeptide. The first polypeptide is, for example, a mucin polypeptide such as PSGL-1 or an alpha glycoprotein such as alpha 1 acid glycoprotein (orosomucoid). The second polypeptide comprises at least a region of an Ig polypeptide. The MA fusion polypeptide is a multimer or preferably a dimer. Also included in the invention is a nucleic acid encoding an MA fusion polypeptide, as well as a vector containing MA fusion polypeptide-encoding nucleic acids described herein, and a cell containing the vectors or nucleic acids described herein.

L9 ANSWER 3 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:129145 CAPLUS
DOCUMENT NUMBER: 138:186310
TITLE: Cutting Edge: Carbohydrate profiling identifies new pathogens that interact with dendritic cell-specific ICAM-3-grabbing nonintegrin on dendritic cells
AUTHOR(S): Appelmelk, Ben J.; van Die, Irma; van Vliet, Sandra J.; Vandenbroucke-Grauls, Christina M. J. E.; Geijtenbeek, Teunis B. H.; van Kooyk, Yvette
CORPORATE SOURCE: Department of Medical Microbiology, Vrije Universiteit Medical Center, Amsterdam, Neth.
SOURCE: Journal of Immunology (2003), 170(4), 1635-1639
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Dendritic cells (DC) are instrumental in handling pathogens for processing and presentation to T cells, thus eliciting an appropriate immune response. C-type lectins expressed by DC function as pathogen-recognition receptors; yet their specificity for carbohydrate structures on pathogens is not fully understood. In this study, the authors analyzed the carbohydrate specificity of DC-specific ICAM-3-grabbing nonintegrin (SIGN)/CD209, the recently documented HIV-1 receptor on DC. The authors' studies show that DC-SIGN binds with high affinity to both synthetic mannose- and fucose-containing glycoconjugates. These carbohydrate structures are abundantly expressed by pathogens as demonstrated by the affinity of DC-SIGN for natural surface glycans of the human pathogens Mycobacterium tuberculosis, **Helicobacter** pylori, Leishmania mexicana, and Schistosoma mansoni. This anal. expands the authors' knowledge on the carbohydrate and pathogen-specificity of DC-SIGN and identifies this lectin to be central in pathogen-DC interactions.
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 22 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003223883 EMBASE
TITLE: The dendritic cell-specific C-type lectin DC-SIGN is a receptor for Schistosoma mansoni egg antigens and recognizes the glycan antigen Lewis x.
AUTHOR: van Die I.; van Vliet S.J.; Nyame A.K.; Cummings R.D.; Bank C.M.C.; Appelmelk B.; Geijtenbeek T.B.H.; van Kooyk Y.
CORPORATE SOURCE: I. van Die, Department of Molecular Cell Biology, VU University Medical Center, Van der Boechorststraat 7, 1081 BT Amsterdam, Netherlands. im.van_die.medchem@med.vu.nl
SOURCE: Glycobiology, (1 Jun 2003) 13/6 (471-478).
Refs: 49
ISSN: 0959-6658 CODEN: GLYCE3
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Schistosoma mansoni soluble egg antigens (SEAs) are crucially involved in modulating the host immune response to infection by S. mansoni. We report

that human dendritic cells bind SEAs through the C-type lectin dendritic cell-specific ICAM-3-grabbing nonintegrin (DC-SIGN). Monoclonal antibodies against the carbohydrate antigens Lewis(x) (Le(x)) and GalNAcβ1-4(Fucα1-3 GlcNAc (LDNF) inhibit binding of DC-SIGN to SEAs, suggesting that these glycan antigens may be critically involved in binding. In a solid-phase adhesion assay, DC-SIGN-Fc binds polyvalent neoglycoconjugates that contain the Le(x) antigen, whereas no binding was observed to Galβ1-4GlcNAc, and binding to neoglycoconjugates containing only α-fucose or oligosaccharides with a terminal α1-2-linked fucose is low. These data indicate that binding of DC-SIGN to Le(x) antigen is fucose-dependent and that adjacent monosaccharides and/or the anomeric linkage of the fucose are important for binding activity. Previous studies have shown that DC-SIGN binds HIV gp120 that contains high-mannose-type N-glycans. Site-directed mutagenesis within the carbohydrate recognition domain (CRD) of DC-SIGN demonstrates that amino acids E(324) and E(347) are involved in binding to HIV gp120, Le(x), and SEAs. By contrast, mutation of amino acid Val(351) abrogates binding to SEAs and Le(x) but not HIV gp120. These data suggest that DC-SIGN recognizes these ligands through different (but overlapping) regions within its CRD. Our data imply that DC-SIGN not only is a pathogen receptor for HIV gp120 but may also function in pathogen recognition by interaction with the carbohydrate antigens Le(x) and possibly LDNF, which are found on important human pathogens, such as schistosomes and the bacterium **Helicobacter** pylori.

L9 ANSWER 5 OF 22 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2003047305 EMBASE
TITLE: Expression of Lewis(b) blood group antigen in **Helicobacter** pylori does not interfere with bacterial adhesion property.
AUTHOR: Zheng P.-Y.; Hua J.; Ng H.-C.; Yeoh K.-G.; Bow H.
CORPORATE SOURCE: Dr. P.-Y. Zheng, Div. of Gastroenterology/Nutrition, Hospital for Sick Children, University of Toronto, 555 University Ave., Toronto, Ont. M5G 1X8, Singapore. pengyuan.zheng@sickkids.ca
SOURCE: World Journal of Gastroenterology, (15 Jan 2003) 9/1 (122-124).
Refs: 19
ISSN: 1007-9327 CODEN: WJGAF2
COUNTRY: China
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Aim: The finding that some **Helicobacter** pylori strains express Lewis b (Le(b)) blood group antigen casts a doubt on the role of Le(b) of human gastric epithelium being a receptor for **H. pylori** . The aim of this study was to determine if expression of Le(b) in **H. pylori** interferes with bacterial adhesion property. Methods: Bacterial adhesion to immobilized Le(b) on microtitre plate was performed in 63 **H. pylori** strains obtained from Singapore using in vitro adherence assay. Expression of Lewis blood group antigens was determined by ELISA assay. Results: Among 63 **H. pylori** strains, 28 expressed Le(b) antigen. In vitro adhesion assay showed that 78.6 % (22/28) of Le(b)-positive and 74.3 % (26/35) of Le(b)-negative **H. pylori** isolates were positive for adhesion to immobilized Le(b) coated on microtitre plate (P=0.772). In addition, blocking of **H. pylori** Le(b) by prior incubation with anti-Le(b) monoclonal antibody did not alter the binding of the bacteria to solid-phase coated Le(b). Conclusion: The present study suggests that expression of Le(b) in **H. pylori** does not interfere with the bacterial adhesion property. This result supports the notion that Le(b) present on human gastric epithelial cells is capable of being a receptor for **H. pylori**.

L9 ANSWER 6 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2003:568523 BIOSIS
DOCUMENT NUMBER: PREV200300563383
TITLE: NO RELATIONSHIP BETWEEN **HELICOBACTER** PYLORI ADHERENCE FACTORS, BABA2, SABA AND GM1 AND GASTRODUODENAL DISEASE.
AUTHOR(S): Kidd, Mark [Reprint Author]; Bourgeois, D. L.; Lastovica, Albert J.; Louw, Japie A.; Sack, David A.
CORPORATE SOURCE: New Haven, CT, USA
SOURCE: Digestive Disease Week Abstracts and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. M895. e-file.

Meeting Info.: Digestive Disease 2003. FL, Orlando, USA. May 17-22, 2003. American Association for the Study of Liver Diseases; American Gastroenterological Association; American Society for Gastrointestinal Endoscopy; Society for Surgery of the Alimentary Tract.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Dec 2003
Last Updated on STN: 3 Dec 2003

AB Introduction: **Helicobacter** pylori virulence and adherence factors play an important role in the development of specific gastric diseases. Studies have demonstrated the clinical relevance of the blood group Ag-binding adhesin, BabA, encoded by babA2. More recently, the SabA adhesin which binds **sialyl-Lewis x** antigens has been identified as potentially of clinical interest. Our group has identified the presence of surface gangliosides (Gm1) in **H. pylori** lipopolysaccharide which binds the cholera toxin receptor in mammalian cells (J. Clin Micro, 1998; 36:2043-5). We postulated that gastroduodenal disease severity would be related to the expression of these bacterial adherence parameters. Methods: Sixty strains isolated from 52 dyspeptic patients (14 with peptic ulcer disease (PUD), 14 with gastric adenocarcinoma (GCA) and 24 with non-ulcer dyspepsia (NUD)) with known virulence gene profiles (cagA, vacA) were examined for the presence of the babA2 and sabA genes using polymerase chain reaction and for the presence of Gm1 binging using an established microtiter enzyme-linked immunosorbent assay. Results: BabA2 was identified in 48% of all strains, and was significantly expressed in strains from NUD (63%, $p < 0.05$, O.R. = 3.74) compared to strains from GCA patients (31%), but not different to PUD strains (41%). In contrast, sabA was identified in all strains irrespective of disease pathology. Gm1 was identified in 82% of strains and was not significantly distributed between the different groups (Chi-square = 2.6, $p = 0.27$). Similar numbers of strains where Gm1+/babA2+ (43%) and Gm1+/babA2- (38%). Significantly more strains from NUD patients were Gm1-/babA2- (22%) compared to GCA strains (0%, $p < 0.05$). No correlation was, however, noted between adherence factors and virulence genes. Conclusion: The babA2 and sabA genes do not correlate with disease severity. Specifically, there was no relationship between babA2 and gastric cancer isolates. While most strains expressed Gm1, a proportion of strains from patients with NUD did not exhibit the Gm1 epitope and were babA2-negative. This study does not support a major role of adherence factors in the development of gastroduodenal disease..

L9 ANSWER 7 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:270559 BIOSIS
DOCUMENT NUMBER: PREV200300270559
TITLE: Recombinant fusion proteins carrying **sialyl-Lewis X** as inhibitors of **Helicobacter** pylori adhesion.
AUTHOR(S): Lofling, Jonas [Reprint Author]; Wreiber, Karin; Engstrand, Lars; Holgersson, Jan
CORPORATE SOURCE: Department of Microbiology, Pathology and Immunologi, Karolinska Institutet, Division of Clinical Immunology, F79, Stockholm, Huddinge, 14186 Stockholm, Sweden jonas.lofling@impi.ki.se; karin.wreiber@smi.ki.se; lars.engstrand@smi.ki.se; jan.holgersson@impi.ki.se
SOURCE: FASEB Journal, (March 2003) Vol. 17, No. 4-5, pp. Abstract No. 627.4. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. San Diego, CA, USA. April 11-15, 2003. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003

AB **Helicobacter** pylori (Hp) is a gram-negative, human pathogen that has been associated with gastric ulcer disease, as well as adenocarcinoma of the stomach. A prerequisite for bacterial infection is attachment, mediated by adhesins on the bacterium often binding to carbohydrates on the target cell. One important carbohydrate determinant supporting Hp adhesion is the **sialyl-Lewis X** (SLex) epitope. In order to assess the ability of SLex-carrying glycoproteins to bind to Hp and prevent infection, we have produced recombinant proteins by on the cDNA level fusing the extracellular domains of proteins known to be highly glycosylated, alpha-1-acid glycoprotein (AGP) or P-selectin glycoprotein ligand-1 (PSGL-1), with the Fc portion of mouse IgG. These

constructs were transfected into 293T, CHO and COS cells together with different cDNAs encoding (1,3-fucosyltransferases. SLex-substituted PSGL-1/IgG could be made in 293T and COS, but not in CHO cells (as expected, because of their lack of O-linked polylactosamine sequences). Interestingly, more SLex was made in COS than in 293T using fucosyltransferase III (FucT-III). N-linked SLex could be made on AGP/IgG in CHO using FucT-VI, but not in COS or 293T cells. Instead 293T cells produced SLex on AGP/IgG when using FucT-VII, whereas we did not detect any SLex-epitopes on AGP/IgG made in COS cells. PSGL-1/IgG made with FucT-VII in 293T cells were shown to strongly bind SLex-binding but not to non-SLex-binding strains of Hp. Further studies are needed in order to assess the ability of these fusion proteins to inhibit binding of Hp to gastric epithelium.

L9 ANSWER 8 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:270709 BIOSIS
DOCUMENT NUMBER: PREV200300270709
TITLE: **Helicobacter** pylori binding to human and rhesus monkey gastric mucins and host changes after inoculation.
AUTHOR(S): Linden, Sara Katarina [Reprint Author]; Mahdavi, Jafar; Hurtig, Marina; Boren, Thomas; Dubois, Andre; Carlstedt, Ingemar
CORPORATE SOURCE: Cell- and Molecular Biology, Lund University, BMC/C13, Lund, 22184, Sweden
sara.linden@medkem.lu.se; jafar.mahdavi@odont.umu.se; marina.hurtig@odont.umu.se; thomas.boren@odont.umu.se; adubois@usuhs.mil; ingemar.carlstedt@medkem.lu.se
SOURCE: FASEB Journal, (March 2003) Vol. 17, No. 4-5, pp. Abstract No. 363.6. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. San Diego, CA, USA. April 11-15, 2003. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003

AB At neutral pH, HP strains expressing BabA adhesins bound to the MUC5AC mucin and to smaller molecules, possibly MUC1, in individuals expressing Lewis b (Leb). In addition, Leb -positive MUC5AC glycoforms differed in their receptor properties for different BabA-positive **H. pylori** strains (Linden et al, Gastroenterology, 2002, in press). At pH 3, Leb -mediated binding was abolished and all strains bound to a putative monomeric mucin of higher charge and larger size than subunits of MUC5AC/MUC6 as well as to a highly charged MUC5AC glycoform. Gastric mucins from rhesus monkey and man were similar with respect to structure, density, carbohydrate compositions and HP binding. Expression of MUC5AC, MUC6, MUC5B, MUC2, Lea, sialyl-Lea, sulfo-Lea, Leb, Lex, sialyl-Lex and Ley were determined after inoculating rhesus monkey with HP. Of the observed changes, an increase of the sialylated antigens were the most prominent finding. The expression of the sialylated antigens increased as early as one week after inoculation and in most cases returned to baseline levels before 10 months (even in the presence of persistent infection).Conclusions: All HP strains investigated have similar binding properties at acidic pH, whereas interactions with human healthy mucins at neutral pH are dependent on the bacterial BabA adhesin and on the host mucin Leb determinant. The host (rhesus monkey model) responds quickly to bacterial challenge by temporarily producing more sialyl-Lex and sialyl-Lea. Bacterial challenge of mucosal surfaces may thus trigger complex transient changes in host glycosyl transferase expression in mucus producing cells and this rapid response will certainly influence the structure of putative colonization targets.

L9 ANSWER 9 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:658147 CAPLUS
DOCUMENT NUMBER: 137:198237
TITLE: Potential use of **Helicobacter** pylori sialic acid binding adhesin gene in diagnosis and treatment of infection
INVENTOR(S): Boren, Thomas; Hammarstroem, Lennart
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002066502	A1	20020829	WO 2002-SE301	20020221

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-269889P P 20010221

AB An isolated **Helicobacter** pylori protein binding to **sialyl-Lewis x** antigen and having an approx. mol. weight of 66kDa and **sialyl-Lewis x** antigen-binding **H.pylori** alleles of the protein, recombinant forms of the protein or the protein alleles, and **sialyl-Lewis x** antigen binding portions of the proteins, are disclosed. The protein or portion of protein maybe used as a medicament or diagnostic antigen, and can be used in a method of determining the presence of **sialyl-Lewis x** antigen-binding **H.pylori** bacteria in a biol. sample. Further, a DNA mol. encoding the protein or portion of protein, a vector comprising the DNA mol., and a host transformed with the vector are comprised by the disclosure. Addnl., a method of determining the presence of **sialyl-Lewis x** or related carbohydrate structures in a sample, is described. This method has a wide range of different applications.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 10 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 2002:314468 CAPLUS

DOCUMENT NUMBER: 136:324173

TITLE: Chimeric genes encoding enzymes for biosynthesis of GDP-L-fucose and fucosylated glycans from GDP-D-mannose for treatment of infections and inflammation

INVENTOR(S): Renkonen, Risto; Mattila, Pirkko; Hirvas, Laura; Hortling, Solveig; Kallioinen, Tuula; Kauranen, Sirkka-liisa; Jaervinen, Nina; Maeki, Minna; Niittymaeki, Jaana; Raebinae, Jarkko

PATENT ASSIGNEE(S): Medicel Oy, Finland

SOURCE: Eur. Pat. Appl., 28 pp.
CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1199364	A2	20020424	EP 2001-660180	20010925
EP 1199364	A3	20040324		
FI 2000002114	A	20020327	FI 2000-2114	20000926
US 2002058313	A1	20020516	US 2001-962805	20010926

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: FI 2000-2114 A 20000926

AB Use of recombinant enzymes for the preparation of GDP-L-fucose and fucosylated glycans is disclosed. GDP-L-fucose functions as a fucose donor in the biosynthetic route leading to the fucosylated glycans, which have therapeutic utility. A process for preparing GDP-L-fucose and fucosylated glycans, and means useful in the process are provided. Said means include enzymes, chimeric enzymes, DNA sequences, genes, vectors and host cells. Fucosylation of glycans on glycoproteins and -lipids requires the enzymic activity of relevant fucosyltransferases and GDP-L-fucose as the donor. Due to the biol. importance of fucosylated glycans, a readily accessible source of GDP-L-fucose would be required. Here the authors describe the construction of a stable recombinant S.cerevisiae strain expressing the E.coli genes gmd and wcaG encoding the two enzymes, GDP-mannose-4,6-dehydratase (GMD) and GDP-4-keto-6-deoxy-D-mannose-3,5-epimerase/4-reductase (GFS) resp., needed to convert GDP-mannose to GDP-fucose via the de novo pathway. Taking advantage of the rich inherent cytosolic GDP-mannose pool in S.cerevisiae cells the authors produced 0.2 mg/l of

GDP-L-fucose with the recombinant yeast strain without addition of any external GDP-mannose. The GDP-L-fucose product may be used as the fucose donor for α-1,3-fucosyltransferase to synthesize **sialyl Lewis x** (sLex), a glycan crucial for the selectin-dependent leukocyte traffic. GDP-L-fucose may also be prepared using the salvage pathway from L-fucose by fucokinase (FK) and GDP-fucose-pyrophosphorylase (PP), synthesized from a chimeric gene. Two rapid and simple procedures for the quant. anal. of GDP-L-fucose (GDP-Fuc) are described. The methods are based on time-resolved fluorescence and microplate assay technol. The first assay relies on measuring the enzyme activity of α-1,3-fucosyltransferase. In this assay, transfer of fucose from GDP-Fuc converts sialyllactosamine to **sialyl Lewis x** tetrasaccharide, which is detected and quantified by relevant antibodies on a microplate. The formation of the reaction product is directly dependent on the presence of GDP-Fuc in the concentration range of 10-10,000 nM. In the second method GDP-Fuc inhibits the binding of fucose-specific Aleuria aurantia lectin to fucosylated glycan on a microwell. The lectin-based assay is less sensitive than the enzyme assay, but it is cheaper and faster. The authors used these assays in monitoring the amount of GDP-Fuc in crude lysates of transgenic yeast, which expresses the enzymes producing GDP-Fuc. The newly developed assays are versatile and applicable to measure also other nucleotide sugars or glycosyltransferase activities in a high-throughput manner.

L9 ANSWER 11 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:30268 BIOSIS
DOCUMENT NUMBER: PREV200300030268
TITLE: Recombinant fusion proteins carrying **sialyl-Lewis X** as inhibitors of **Helicobacter** pylori adhesion.
AUTHOR(S): Lofling, Jonas [Reprint Author]; Wreiber, Karin; Falk, Per; Engstrand, Lars; Holgersson, Jan [Reprint Author]
CORPORATE SOURCE: Department of Microbiology, Pathology and Immunology, Karolinska Institutet, Stockholm, Sweden
SOURCE: Glycobiology, (October 2002) Vol. 12, No. 10, pp. 663. print.
Meeting Info.: 7th Annual Conference of the Society for Glycobiology. Boston, MA, USA. November 09-12, 2002. Society for Glycobiology.
ISSN: 0959-6658.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Jan 2003
Last Updated on STN: 8 Jan 2003

L9 ANSWER 12 OF 22 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002394294 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12142529
TITLE: **Helicobacter** pylori SabA adhesin in persistent infection and chronic inflammation.
AUTHOR: Mahdavi Jafar; Sonden Berit; Hurtig Marina; Olfat Farzad O; Forsberg Lina; Roche Niamh; Angstrom Jonas; Larsson Thomas; Teneberg Susann; Karlsson Karl-Anders; Altraja Siiri; Wadstrom Torkel; Kersulyte Dangeruta; Berg Douglas E; Dubois Andre; Petersson Christoffer; Magnusson Karl-Eric; Norberg Thomas; Lindh Frank; Lundskog Bertil B; Arnqvist Anna; Hammarstrom Lennart; Boren Thomas
CORPORATE SOURCE: Department of Odontology/Oral Microbiology, Umea University, SE-901 87 Umea, Sweden.
CONTRACT NUMBER: P30 DK52574 (NIDDK)
RO1 AI38166 (NIAID)
RO1 DK53727 (NIDDK)
RO3 AI49161 (NIAID)
SOURCE: Science, (2002 Jul 26) 297 (5581) 573-8.
Journal code: 0404511. ISSN: 1095-9203.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020727
Last Updated on STN: 20020821
Entered Medline: 20020820
AB **Helicobacter** pylori adherence in the human gastric mucosa involves specific bacterial adhesins and cognate host receptors. Here, we identify sialyl-dimeric-Lewis x glycosphingolipid as a receptor for **H. pylori** and show that **H. pylori**

infection induced formation of **sialyl-Lewis x** antigens in gastric epithelium in humans and in a Rhesus monkey. The corresponding sialic acid-binding adhesin (SabA) was isolated with the "retagging" method, and the underlying sabA gene (JHP662/HP0725) was identified. The ability of many **H. pylori** strains to adhere to sialylated glycoconjugates expressed during chronic inflammation might thus contribute to virulence and the extraordinary chronicity of **H. pylori** infection.

L9 ANSWER 13 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:586389 BIOSIS
DOCUMENT NUMBER: PREV200200586389
TITLE: Virulence factors, cagA and vacA, and Lewis antigen expression in **Helicobacter** pylori isolates from Spanish paediatric patients.
AUTHOR(S): Alarcon, T. [Reprint author]; Garcia-Campos, J. A. [Reprint author]; Moran, A. P.; Domingo, D. [Reprint author]; Diaz-Reganon, J. [Reprint author]; Martinez, M. J.; Lopez-Brea, M. [Reprint author]
CORPORATE SOURCE: Hosp. Univ. de la Princesa, Madrid, Spain
SOURCE: Gut, (September, 2002) Vol. 51, No. Supplement 2, pp. A15-A16. print.
Meeting Info.: XVth International Workshop on Gastrointestinal Pathology and Helicobacter. Athens, Greece. September 11-14, 2002.
CODEN: GUTTAK. ISSN: 0017-5749.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002

L9 ANSWER 14 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:586332 BIOSIS
DOCUMENT NUMBER: PREV200200586332
TITLE: Serological and structural characterization of **Helicobacter** bizozzeronii lipopolysaccharide.
AUTHOR(S): Moran, A. P. [Reprint author]; Ferris, J. A. [Reprint author]; Kocharova, N. A.; Knirel, Y. A.; Widmalm, G.; Andersen, L. P.; Jansson, P. E.
CORPORATE SOURCE: National University of Ireland, Galway, Galway, Ireland
SOURCE: Gut, (September, 2002) Vol. 51, No. Supplement 2, pp. A1. print.
Meeting Info.: XVth International Workshop on Gastrointestinal Pathology and Helicobacter. Athens, Greece. September 11-14, 2002.
CODEN: GUTTAK. ISSN: 0017-5749.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002

L9 ANSWER 15 OF 22 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001691564 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11737200
TITLE: Cloning and expression of **Helicobacter** pylori GDP-l-fucose synthesizing enzymes (GMD and GMER) in Saccharomyces cerevisiae.
AUTHOR: Jarvinen N; Maki M; Rabina J; Roos C; Mattila P; Renkonen R
CORPORATE SOURCE: Department of Bacteriology and Immunology, Haartman Institute and Biomedicum, University of Helsinki, Finland.
SOURCE: European journal of biochemistry / FEBS, (2001 Dec) 268 (24) 6458-64.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011213
Last Updated on STN: 20020125
Entered Medline: 20020115
AB **Helicobacter** pylori is a Gram-negative gastric pathogen causing diseases from mild gastric infections to gastric cancer. The difference in clinical outcome has been suggested to be due to strain differences. **H. pylori** undergoes phase variation by changing its lipopolysaccharide structure according to the environmental conditions.

The O-antigen of **H. pylori** contains fucosylated glycans, similar to Lewis structures found in human gastric epithelium. These Lewis glycans of **H. pylori** have been suggested to play a role in pathogenesis in the adhesion of the bacterium to gastric epithelium. In the synthesis of fucosylated structures, GDP-l-fucose is needed as a fucose donor. Here, we cloned the two key enzymes of GDP-l-fucose synthesis, **H. pylori** gmd coding for GDP-d-mannose dehydratase (GMD), and gmer coding for GDP-4-keto-6-deoxy-d-mannose-3,5-epimerase/4-reductase (GMER) and expressed them in an enzymatically active form in Saccharomyces cerevisiae. The end product of these enzymes, GDP-l-fucose was used as a fucose donor in a fucosyltransferase assay converting sialyl-N-acetyllactosamine to **sialyl Lewis X**.

L9 ANSWER 16 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:688099 CAPLUS
DOCUMENT NUMBER: 133:276347
TITLE: Use of fucosylated sialylated N-acetyllactosamine carbohydrate structures for inhibition of bacterial adherence and treatment of conditions related to infection by **Helicobacter** pylori and related gastrointestinal pathogens
INVENTOR(S): Boren, Thomas; Hammarstrom, Lennart; Karlsson, Karl-Anders; Teneberg, Susann
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000056343	A1	20000928	WO 2000-SE514	20000316
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1169044	A1	20020109	EP 2000-921217	20000316
EP 1169044	B1	20030910		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002539266	T2	20021119	JP 2000-606247	20000316
AT 249227	E	20030915	AT 2000-921217	20000316

PRIORITY APPLN. INFO.: SE 1999-1007 A 19990319
WO 2000-SE514 W 20000316

AB A fucosylated sialylated N-acetyllactosamine structure such as a sialyl-Lewis antigen carbohydrate structure, for example **sialyl-Lewis x** and in particular dimeric or repetitive **sialyl-Lewis x**, can be used for the preparation of a pharmaceutical composition for the treatment or prophylaxis in humans of conditions involving infection by **Helicobacter** pylori and related pathogens of the human gastrointestinal mucosa. Further, the conditions can be treated through the administration of a fucosylated sialylated lactosamine structure, such as a sialyl-Lewis antigen carbohydrate structure, or corresponding antibodies, to patients in need thereof.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 17 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:847075 CAPLUS
DOCUMENT NUMBER: 134:129472
TITLE: Inhibition of nonopsonic **Helicobacter** pylori-induced activation of human neutrophils by sialylated oligosaccharides
AUTHOR(S): Teneberg, Susann; Jurstrand, Margaretha; Karlsson, Karl-Anders; Danielsson, Dan
CORPORATE SOURCE: Institute of Medical Biochemistry, Goteborg University, Goteborg, SE 405 30, Swed.
SOURCE: Glycobiology (2000), 10(11), 1171-1181
CODEN: GLYCE3; ISSN: 0959-6658

PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Certain strains of **Helicobacter** pylori have nonopsonic neutrophil-activating capacity. Some **H.pylori** strains and the neutrophil-activating protein of **H.pylori** (HPNAP) bind selectively to gangliosides of human neutrophils. To determine if there is a relationship between the neutrophil-activating capacity and the ganglioside-binding ability, a number of **H.pylori** strains, and HPNAP, were incubated with oligosaccharides, and the effects on the oxidative burst of subsequently challenged neutrophils was measured by chemiluminescence and flow cytometry. Both by chemiluminescence and flow cytometry a reduced response was obtained by incubation of **H .pylori** with sialic acid-terminated oligosaccharides, whereas lactose had no effect. The redns. obtained with different sialylated oligosaccharides varied to some extent between the **H. pylori** strains, but in general 3'-sialyllactosamine was the most efficient inhibitor. Challenge of neutrophils with HPNAP gave no response in the chemiluminescence assay, and a delayed moderate response with flow cytometry. Preincubation of the protein with 3'-sialyllactosamine gave a slight reduction of the response, while 3'-sialyllactose had no effect. The current results suggest that the nonopsonic **H.pylori** -induced activation of neutrophils occurs by lectinophagocytosis, the recognition of sialylated glycoconjugates on the neutrophil cell surface by a bacterial adhesin leads to phagocytosis and an oxidative burst with the production of reactive oxygen metabolites.
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 18 OF 22 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000098506 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10632700
TITLE: Lipopolysaccharide structures of **Helicobacter** pylori genomic strains 26695 and J99, mouse model **H . pylori** Sydney strain, **H. pylori** P466 carrying **sialyl Lewis X**, and **H. pylori** UA915 expressing Lewis B classification of **H. pylori** lipopolysaccharides into glycotype families.
AUTHOR: Monteiro M A; Appelmelk B J; Rasko D A; Moran A P; Hynes S O; MacLean L L; Chan K H; Michael F S; Logan S M; O'Rourke J; Lee A; Taylor D E; Perry M B
CORPORATE SOURCE: Institute for Biological Sciences, National Research Council, Ontario, Canada.. Mario.Monteiro@nrc.ca
SOURCE: European journal of biochemistry / FEBS, (2000 Jan) 267 (2) 305-20.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000320
Last Updated on STN: 20000320
Entered Medline: 20000307
AB This study describes the molecular makeup of the cell-wall lipopolysaccharides (LPSs) (O-chain polysaccharide-->core oligosaccharide-->lipid A) from five **Helicobacter** pylori strains: **H. pylori** 26695 and J99, the complete genome sequences of which have been published, the established mouse model Sydney strain (SS1), and the symptomatic strains P466 and UA915. All chemical and serological experiments were performed on the intact LPSs. **H . pylori** 26695 and SS1 possessed either a low-Mr semi-rough-form LPS carrying mostly a single Ley type-2 blood-group determinant in the O-chain region covalently attached to the core oligosaccharide or a high-Mr smooth-form LPS, as did strain J99, with an elongated partially fucosylated type-2 N-acetyllactosamine (polyLacNAc) O-chain polymer, terminated mainly by a Lex blood-group determinant, connected to the core oligosaccharide. In the midst of semi-rough-form LPS glycoforms, **H. pylori** 26695 and SS1 also expressed in the O-chain region a difucosylated antigen, alpha-L-Fucp(1-3)-alpha-L-Fucp(1-4)-beta-D-GlcpNAc, and the cancer-cell-related type-1 or type-2 linear B-blood-group antigen, alpha-D-Galp(1-3)-beta-D-Galp(1-3 or 4)-beta-D-GlcpNAc. The LPS of **H. pylori** strain P466 carried the cancer-associated type-2 sialyl Lex blood-group antigen, and the LPS from strain UA915 expressed a type-1 Leb blood-group unit. These findings should aid investigations that focus on identifying and characterizing genes responsible for LPS biosynthesis in genomic strains 26695 and J99, and in

understanding the role of **H. pylori** LPS in animal model studies. The LPSs from the **H. pylori** strains studied to date were grouped into specific glycotype families.

L9 ANSWER 19 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:267188 BIOSIS
DOCUMENT NUMBER: PREV200000267188
TITLE: Defining the roles of lymphocytes and **sialyl-Lewis-X** (sLex) in **Helicobacter** in induced gastric injury.
AUTHOR(S): Beck, Paul L. [Reprint author]; Xavier, Ramnik J.; Kosaka, Takeo; Dangler, Charles A.; Wang, Timothy C.; Fox, James G.
CORPORATE SOURCE: GI Research Group, Univ of Calgary, Calgary, AB, Canada
SOURCE: Gastroenterology, (April, 2000) Vol. 118, No. 4 Suppl. 2 Part 1, pp. AGA A737. print.
Meeting Info.: 101st Annual Meeting of the American Gastroenterological Association and the Digestive Disease Week. San Diego, California, USA. May 21-24, 2000. American Gastroenterological Association.
CODEN: GASTAB. ISSN: 0016-5085.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jun 2000
Last Updated on STN: 5 Jan 2002

L9 ANSWER 20 OF 22 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:486706 BIOSIS
DOCUMENT NUMBER: PREV199900486706
TITLE: **Helicobacter** pylori attaches to NeuAcalpha2,3Galbeta1,4 glycoconjugates, including the tumor associated antigen sialyl-Lewisx, produced in the gastric epithelium of transgenic mice lacking parietal cells.
AUTHOR(S): Guruge, J. L. [Reprint author]; Syder, A. J. [Reprint author]; Lorenz, R. G. [Reprint author]; Falk, P. G. [Reprint author]; Gordon, J. I. [Reprint author]
CORPORATE SOURCE: Dept. of Mol. Biol. and Pharm., Washington Univ., St. Louis, MO, 63110, USA
SOURCE: Gut, (Sept., 1999) Vol. 45, No. SUPPL. 3, pp. A35. print.
Meeting Info.: XIIth International Workshop on Gastroduodenal Pathology and Helicobacter pylori. Helsinki, Finland. September 2-4, 1999.
CODEN: GUTTAK. ISSN: 0017-5749.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 1999
Last Updated on STN: 16 Nov 1999

L9 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:806793 CAPLUS
DOCUMENT NUMBER: 130:62948
TITLE: α1,3-fucosyltransferase of **Helicobacter** pylori and its use for oligosaccharide synthesis
INVENTOR(S): Taylor, Diane E.; Ge, Zhongming
PATENT ASSIGNEE(S): The Governors of the University of Alberta, Can.
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9855630	A2	19981210	WO 1998-CA564	19980605
WO 9855630	A3	19990304		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9880050	A1	19981221	AU 1998-80050	19980605
US 6399337	B1	20020604	US 1998-92315	19980605

US 2002068347	A1	20020606	US 2000-733524		20001207
US 6534298	B2	20030318			
US 2002164749	A1	20021107	US 2002-120319		20020409
US 2003166211	A1	20030904	US 2002-189977		20020703
US 2003166212	A1	20030904	US 2003-392098		20030317
PRIORITY APPLN. INFO.:			US 1997-48857P	P	19970606
			US 1998-92315	A3	19980605
			WO 1998-CA564	W	19980605
			US 2000-733524	A1	20001207
			US 2002-120319	A1	20020409

AB A bacterial α1,3-fucosyltransferase gene and deduced amino acid sequence is provided from **Helicobacter** pylori. An unusual feature of the open reading frame is the presence of 8 direct repeats of 21 nucleotides (7 amino acid repeats proximal to the C-terminus). The amino acid sequence is highly conserved except for the repeat regions. The gene is useful for preparing α1,3-fucosyltransferase polypeptide, and active fragment thereof, which can be used in the production of oligosaccharides such as Lewis X, Lewis Y, and **sialyl Lewis X**, which are structurally similar to certain tumor-associated carbohydrate antigens found in mammals. These product glycoconjugates also have research and diagnostic utility in the development of assays to detect mammalian tumors. In addition the polypeptide of the invention can be used to develop diagnostic and research assays to determine the presence of **H. pylori** in human specimens.

L9 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:612015 CAPLUS
DOCUMENT NUMBER: 129:229679
TITLE: Sialyl lewis antigens as targets for immunotherapy
INVENTOR(S): Ravindranath, Mepur H.; Morton, Donald L.
PATENT ASSIGNEE(S): John Wayne Cancer Institute, USA
SOURCE: PCT Int. Appl., 126 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9839027	A2	19980911	WO 1998-US4314	19980305
WO 9839027	A3	19990107		
W: AU, CA, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9865428	A1	19980922	AU 1998-65428	19980305
PRIORITY APPLN. INFO.:			US 1997-811281	19970305
			WO 1998-US4314	19980305

AB Sialyls Lewis (sLe) antigens are functionally important, immunogenic, tumorigenic or differentiation antigens and potential targets for both passive and active specific immunotherapy of melanoma and other cancers sharing these antigens. The present invention concerns the use of such antigens in vaccine formulations for the treatment of a variety of cancers and in particular melanoma. The B lymphocytes from the vaccine recipients will be used to harvest human monoclonal antibodies and use it as a drug for treatment of melanoma and other cancers.

L16 ANSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 1999065108 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9849856
TITLE: **Helicobacter** pylori infection produces reversible glycosylation changes to gastric mucins.
COMMENT: Comment in: Virchows Arch. 1999 Oct;435(4):458-60. PubMed ID: 10526012
AUTHOR: Ota H; Nakayama J; Momose M; Hayama M; Akamatsu T; Katsuyama T; Graham D Y; Genta R M
CORPORATE SOURCE: Department of Medicine, Veterans Affairs Medical Center and Baylor College of Medicine, Houston, Tex, USA.
SOURCE: Virchows Archiv : an international journal of pathology, (1998 Nov) 433 (5) 419-26.
Journal code: 9423843. ISSN: 0945-6317.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 20000314
Entered Medline: 19981229

AB The protective ability of gastric mucins may depend largely on their oligosaccharide chains. We evaluated the effects of **H. pylori** infection on the glycosylation of gastric mucins. Gastric biopsy specimens from 20 **H. pylori**-infected patients before and after cure of the **H. pylori** infection and 8 normal uninfected volunteers were examined by immunostaining for simple mucin-type glycoproteins and blood-group-related antigens bearing type 1 chain backbone. The immunoreactivity in different gastric compartments was evaluated. Simple mucin-type glycoproteins and blood-group-related antigens were expressed in surface mucous cells. Simple mucin-type glycoproteins showed antrum-predominant expression in normal volunteers and were found in significantly fewer surface mucous cells in infected patients than in normal volunteers; their expression was restored after eradication of **H. pylori**. **Sialyl Lewis(a)** and Lewis(b) were expressed in fewer surface mucous cells after than before eradication. The patterns of glycosylation of gastric mucins vary in different gastric compartments and are reversibly altered by **H. pylori** infection. These alterations may affect the protective functions of gastric mucins.